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JANUARY, 1944

EDITORIAL

THE YEAR AHEAD

ANOTHER year has passed and we have now entered 1944. At this time we extend to our readers the season's greetings, with the hope that when the year ends the world will be in a more peaceful state than we find it now. The veterinary profession in this country has, in the dark days of war, accepted its responsibility for maintaining the health of the livestock. It is true that our knowledge of the best methods of control of disease may be limited in some directions, but if we apply all the reliable proved methods to the best of our ability we shall have gone a long distance in improving animal health and in preventing the spread of infectious disease. In the year ahead the veterinary profession will be called upon to continue and to extend its care of the livestock throughout the country. Livestock is precious and represents much wealth. A perusal of the agricultural Press indicates very clearly the value that is placed on our animals : their health is, to a large extent, in the hands of the veterinary profession. There is also a very definite indication that the agricultural public is becoming more disease-minded and is anxious to maintain its livestock in a healthy state. It looks to the veterinary profession for guidance on the subject. It is our duty, therefore, to give of our best, to extend our knowledge by all the means available and to put into practice the proved methods of treatment and control of the diseases which hamper the livestock industry to such an extent to-day. Every year sees new methods brought into being : 1944 will be no exception. It will be our aim through the publication of original articles and suitable references to publications elsewhere to keep our readers informed of the progress throughout the year.

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General Articles

SOMATIC VARIATION IN SOME KENYA STRAINS OF SALMONELLA

By D. SLAVIN

I HAD noted when working with some local strains of S. typhimurium and S. dublin an occasional marked cross-agglutination with pure O sera prepared from these strains. The agglutination was so pronounced that an explanation other than the presence of the common XII antigen appeared probable. A temporary solution was forthcoming when Kauffmann[1] indicated the presence of the I antigen in S. dublin and showed that this factor was produced irregularly. In a plated culture colonies rich in this antigen and others poor in it or altogether devoid of it could be demonstrated. Some strains of S. typhimurium also contain it. About this time we had been notified of a number of deaths from calf paratyphoid (confirmed by laboratory examination) in calves vaccinated against this disease at or within a few days of birth. As this failure of the vaccine to protect might have been attributable to the said somatic variation, it was resolved to test our vaccine strain for it and subsequently to test the virulence and immunising power of variants with the somatic complex I, IX, and those with the single factor IX. One of our vaccine strains had been isolated from the liver of a calf which had been destroyed for diagnostic purposes in November, 1939. This calf had come from a particularly severe outbreak in which a farmer had lost 70 out of 140 calves in three months. All these animals had been vaccinated with a dose of approximately 2,000 million organisms of another strain given subcutaneously in a single dose. The newly isolated strain was shown to be S. dublin by the following tests (see Table 1).

Technique.—All strains quoted in the tables of this paper were shown to be the species stated by testing them against Kauffmann's sera and testing rabbit sera prepared from them against Kauffmann's type strains. Both type sera and type strains were sent to me by Dr. Kauffmann, of the International Salmonella Centre, Copenhagen, in May, 1939, and I here express my gratitude for his kindness. Only one strain of S. dublin was studied in detail serologically, but over 40 other strains of this species and several of S. gallinarum and S. typhimurium were found to exhibit the type of somatic variation described below. Except where otherwise stated, all tests were done by slide agglutination method, which is now recognised as a reliable diagnostic test. All cultures and sera which came from the International Salmonella Centre are marked with the initial K. All sera not so marked were made by myself. Flagellar antigens, except as stated in table 6, were suspended in 0.5 per cent. phenol saline, and all somatic antigens were made by heating OH suspensions in 70 per cent. alcohol for one hour at 70 degrees C. in a water bath. Sera for slide agglutination tests were diluted 1 in 5 or 1 in 10 according as the titre was 1 in 320 or 1 in 640 respectively. OH sera for slide agglutination tests were diluted 1 in 50 or 1 in 100 according as the titre was 1 in 3,200 or 1 in 6,400. When virulence tests were done the inoculum was shown to be alive by simultaneous culture tests on

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TABLE 1

Typing of S. dublin 7999/39 against Kauffmann's Sera and Type Strain.

Antigen	Serum	Absorbed with	Dilution															
			1/40	1/80	1/160	1/320	1/640	1/1280	1/2560	1/100	1/200	1/400	1/800	1/1600	1/3200	1/6400	1/12800	S.A.
S. dublin	K.6 S. enteritidis (O)		++	++	++	++	++	+	—									+
7999/39 (O)	(I) IX (XII)		++	++	++	++	+											+
Do.	S. dublin 7999/39 (O)	K.64 S. enteritidis (O)	—	—	—	—	—	—	—									—
Do.	Do.		++	++	++	++	++	+	—									+
			++	++	++	++	+	+										+
K.65 S. dublin (O)	Do.		++	++	++	++	++	+	—									+
			++	++	++	++	+											+
S. dublin 7999/39 (OH)	S. dublin 7999/39 (OH)	K.65 S. dublin (O+OH)	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Do.	K.31 S. dublin - - - gp. . .	K.65 S. dublin (O)								++	++	++	++	++	+	—	—	+
										++	++	++	++	++	+			+
Do.	K.31 S. dublin (I) IX (XII) gp.									++	++	++	++	++	++	++	—	+
										++	++	++	++	++	++	+		+
Do.	K.31 S. dublin - - - p. . .	K.65 S. dublin (O) K.64 S. enteritidis (OH)								++	++	++	++	++	+	—	—	+
										++	++	+	+	+	+			+

Test Time: Two hours at 52 deg. Cent. All antigens in 0.5 per cent. carbol saline.
S.A. signifies slide agglutination test.

plain agar plates which were subsequently tested against the requisite sera by slide agglutination test. All sera prepared by me were preserved by adding to them 0.5 per cent. phenol in 50 per cent. glycerin in equal volume. All rabbits used for serum production were shown before inoculation to be free from demonstrable amounts of agglutinins for S. dublin, S. gallinarum and S. typhimurium. The technique of doing the slide agglutination test was as follows: the agar plate was placed on a Pake's enumerating disc and a separate colony was picked off and emulsified on a slide in a drop of serum diluted as above. The number of the square in which the colony was situated was recorded and then the slide was gently heated over a Bunsen flame and rotated from side to side, and examined under the dissecting microscope at 16 magnifications. Complete O agglutination was recorded as + + and minor degrees as + and ±. As pure H agglutination is never a complete clumping of the organisms and clearing of the emulsion, the standard of completeness adopted was the equivalent of that which occurred when one of Kauffmann's type strains was mixed with its homologous pure H absorbed serum. Except where otherwise stated, all sera were used unabsorbed. All cultures were smooth and were made on plain agar, and at no time during these experiments was any substance added which is known to suppress certain antigens. The final concentration of all antigens used in the tube agglutination tests was equivalent to tube 2 in the Burroughs & Wellcome nephelometer scale.

S. dublin 7999/39 was repeatedly replated, and after a number of replatings colonies were found that gave the following reactions by slide agglutination test:

TABLE 2

Slide Agglutination Tests of S. dublin 7999/39 against Unabsorbed K. Sera

Organism		Sera	
		K.1	K.6
		S. paratyphi A (I, II, [XII])	S. enteritidis ([I], IX, [XII])
S. dublin 7999/39 var.	A	+ +	+ +
" " "	B	—	+ +

These variants were then called var. A and var. B, the former being the variant that presumably contained the I antigen in demonstrable amount. It was comparatively easy to obtain colonies which showed moderate O agglutination in the K.1 S. paratyphoid A somatic serum, but it required months of replating before two colonies were isolated, one of which gave rapid and complete agglutination in this serum and one which failed to give any agglutination. This was achieved by inoculating four litre flasks of broth from likely colonies and subsequently replating from these daily for a week on large Petri dishes. These two colonies, var. A and var. B were then subcultivated into Roux flasks which were incubated for 18 hours at 37 degrees C. When tested the next morning both cultures had retained completely the characteristics of the parent colonies, i.e., by slide agglutination tests against K.1 S. paratyphoid A somatic serum, var. A was rapidly agglutinated while var. B failed completely to be agglutinated. The growth was washed off with sterile saline and portions of each were set aside for virulence tests on mice, further portions being carbolised or formalised for use as vaccines and the preparation of sera for tube and slide agglutination tests. Thus, the self-same growths were used throughout the first series of experiments.

First Series of Virulence Tests.—Mice were inoculated intraperitoneally a

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few hours afterwards with live cultures of each variant in doses of approximately 100 to 10,000,000 organisms. The results are shown in Table 3.

TABLE 3

First Series Virulence Tests S. dublin 7999/39 var. B

Approx. No. of Organisms in Inoculum	No. of Mice Inoculated	No. Died	Days of Death after Inoculation	Organisms Recovered	S.A. Reaction Against K. Sera K.1	K.6	K.7
100	5	3	2nd 8th 9th	7999/39 B	—	++	—
1,000	5	3	Do.	Do.	—	++	—
10,000	5	3	2nd (2) 5th	Do.	—	++	—
100,000	5	4	1st 2nd 7th 9th	Do.	—	++	—
1,000,000	5	5	2nd 3rd 4th (2) 8th	Do.	—	++	—
10,000,000	5	5	3rd 4th (2) 5th 6th	Do.	—	++	—

First Series Virulence Tests S. dublin 7999/39 var. A

100	5	0		
1,000	5	0		
10,000	5	0		
100,000	5	1 (a)	28th	Cultures Negative
1,000,000	5	1 (a)	10th	Do.
10,000,000	5	1 (a)	20th	Do.

S.A. indicates slide agglutination test.
Factors in K sera: K.1 S. paratyphi A I II (XII).
K.6 S. enteritidis (I) IX (XII).
K.7 S. typhi Vi (IX) Vi (d).
(a) Cultures from liver on plain agar. No lesions present.
The test survival time was one month.

It will be seen that var. A was completely avirulent while var. B was of considerable virulence. Cultures were made on plain agar from the liver of each mouse carcase as the animal died. These cultures were examined by slide agglutination test after 18 hours' incubation, ten separate colonies being selected from each plate. Large discrete ones were marked and a portion was taken up with a platinum loop and emulsified on a slide with the K.1 S. paratyphi A somatic serum, and the emulsion was examined under the dissecting microscope at 16 magnifications. The needle was sterilised and the same procedure was followed with K.6 S. dublin somatic serum. The heavy initial needle-track growth was tested against K.31 S. dublin OH serum and in all cases reacted positively. A few colonies gave a very slight reaction in the K.1 serum, but almost all were frankly negative. Thus, mice in the var. B series gave cultures true to the parent type in all cases, while the var. A mice that died failed to show any lesions and their cultures were negative for S. dublin, so their cause of death can be regarded as due to intercurrent factors not related to the experiment.

As it was thought at the time that the heightened virulence of var. B might possibly have been due to the presence of a Vi antigen, both variants were tested against an unabsorbed Vi serum, K.7 [(IX) Vi (d)], with the following results:

TABLE 4

Slide Agglutination Reactions of S. dublin 7999/39 Against Unabsorbed K. Sera

Antigen	Sera K.1 I II (XII)	K.6 (I) IX (XII)	K.7 (IX) Vi (d)
S. dublin 7999/39 A (OH)	++	++	++
B	—	++	—
K.57 " S. typhi " H901 (OH) "	—	++	—
K.59 S. typhi V (OH)	—	—	++
VW	—	++	++
K.1 " S. paratyphi A " (OH)	++	+	+
		—	—

That cross agglutination of var. A was not due to the presence of I, II, or Vi was shown when these sera were absorbed with K.2 S. paratyphi A durazzo and K.57 S. typhi H 901 respectively. (Tables 5 and 7.)

TABLE 5

Slide Agglutination Reactions of S. dublin 7999/39 Against Absorbed K. Sera

Antigen	Sera	
	K.1 S. paratyphoid A absorbed	K.7 S. typhi Vi absorbed
	K.2 S. paratyphoid A (durazzo)	K.57 S. typhi H901 (OH)
S. dublin 7999/39 A (OH)	—	—
" B "	—	—
K.59 " S. typhi V (OH) "		++
K.1 S. paratyphi A (OH)	++	

Kauffmann had shown that European strains of S. dublin did possess the I antigen, but in irregular amounts. In the examination of over 40 Kenya strains I have been unable to demonstrate its presence when using Kauffmann's K.1 S. paratyphi A serum absorbed with K.2 S. paratyphi A durazzo, either by slide or by tube agglutination, and I think it is safe to assume that it does not occur or is rare in these strains.

TABLE 6

Serological Tests S. dublin 7999/39 Absorbed Sera

Serum		Absorbed	Antigen	Dilution 1/20	1/40	1/80	1/160	1/320	S.A.
Var. A (OH)	f.	Var. B (O+OH)	Var. A (OH)	++	+	+	—	—	+
				++	+	+			
Var. B (OH)	f.	Var. A (O+OH)	Var. B (OH)	—	—	—	—	—	—
"		"	Var. A "	—	—	—	—	—	—
Var. A (O)	c.	Var. B (O)	"	++	+	+	—	—	+
				++	+	+			
Var. B (O)	c.	Var. A (O)	Var. B (O)	—	—	—	—	—	—
"		"	Var. A (O)	—	—	—	—	—	—
Var. A (O)	c.	Var. B (O)	K.1 S. paratyphi A (O)	+	+		—	—	+
				+	+				
Var. B (O)	c.	Var. A (O)	" "	—	—	—	—	—	—
Var. A (O)	c.	Var. B (O)	S. typhi-murium var. A. (mouse strain) (O)	+	+		—	—	—
				+	+				
"		"	" " " var. B	—	—	—	—	—	—
"		"	S. gallinarum Kabete var. A	++	+				
				++	+	+	—	—	+
"		"	" " var. B	—	—	—	—	—	—

All agglutinations granular. f indicates that the sera were prepared from whole culture suspended in 0.25 per cent. formol saline and that both absorbing and test antigens were also in this. c indicates carbol suspensions.

Tube Agglutination Tests.—Sera were prepared by inoculating rabbits with the remaining antigen left over from the first series of virulence tests. This had been killed by the addition of 0.5 per cent. carbol or 0.25 per cent. formol and was divided into two portions for the preparation of O and OH sera and the carrying out of agglutination tests. Table 6 shows the results of these tests and clearly indicates the presence of a non-specific factor in both the O and OH sera prepared from var. A. By absorption of var. A somatic serum

TABLE 7
Tube Agglutination Tests

TABLE 7
Tube Agglutination Tests

Serum	Antigen	Dilution										
		1/40	1/80	1/160	1/320	1/640	1/100	1/200	1/400	1/800	1/1600	1/3200
K.1 S. paratyphi A	S. dublin 7999/39	++	++	+								
I, II, (XII)	var. A (O)	++	++	+	+	−						
	S. dublin 7999/39											
Do.	var. B (O)	+	−	−	−	−						
K.6 S. enteritidis	S. dublin 7999/39	++	++	++	++	++						
(I), IX, (XII)	var. A (O)	++	++	++	++	++						
	S. dublin 7999/39	++	++	+								
Do.	var. B (O)	++	++	+	−	−						
K.7 S. typhi Vi	S. dublin 7999/39	+										
			+	−	−	−						
(IX), Vi, (d)	var. A (OH)	+										
	S. dublin 7999/39											
Do.	var. B (OH)	−	−	−	−	−						
K.31 S. dublin absorbed	S. dublin 7999/39						++	++	++	++	++	++
K.65 S. dublin (O)	var. A (OH)						++	++	++	++	++	++
	S. dublin 7999/39						++	++	++	++	++	++
Do.	var. B (OH)						++	++	++	++	++	++
K.7 S. typhi Vi	S. dublin 7999/39	+										
(IX), Vi, (d)	var. A (O)	+	−	−	−	−						
	S. dublin 7999/39	−	−	−	−	−						
Do.	var. B (O)											
K.7 S. typhi Vi absorbed		−	−	−	−	−						
K.57 S. typhi H 901	K.57 S. typhi H 901	++	++	++	+							
Do.	K.62 S. typhi Vi	++	++	+	+	+						
	S. dublin 7999/39											
Do.	var. A (O)	−	−	−	−	−						
	S. dublin 7999/39											
Do.	var. A (OH)	−	−	−	−	−						
	S. dublin 7999/39											
Do.	var. B (O)	−	−	−	−	−						
	S. dublin 7999/39											
Do.	var. B (OH)	−	−	−	−	−						
K.1 S. paratyphi A absorbed	S. dublin 7999/39											
K.2 S. paratyphi A	var. A (O)											
var. durazzo		−	−	−	−	−						
	S. dublin 7999/39											
Do.	var. B (O)	−	−	−	−	−						

with a heavy var. B somatis suspension this non-specific factor could be left in apparently pure state. A further series of experiments was made and these confirm the presence of the non-specific factor in var. A sera and the absence of the I and Vi antigens from both variants.

Vaccination Tests.—These were made on only a small number of animals and give no reliable indication of the immunising value of either variant. They are quoted here only because they again show the complete avirulence of var. A and the high virulence of var. B. Twenty mice were vaccinated against each variant by inoculating approximately 200 millions formalin-killed organisms intraperitoneally, followed a week later by twice that dose. The animals were then given approximately 500,000 live organisms of each variant intraperitoneally 14 days after the second vaccinating dose. The results are shown in Table 8. The test time was one month.

TABLE 8

Vaccination Tests

Vaccinated Var. A

Inoculated Var. A				Inoculated Var. B			
No. of mice	No. died	Days of death	Organisms recovered	No. of mice	No. died	Days of death	Organisms recovered
10	2	12th 14th	Cultures sterile	10	4	7th 8th 10th 20th	S. dublin 7999/39 var. B

Vaccinated Var. B

Inoculated Var. A				Inoculated Var. B			
No. of mice	No. died	Days of death	Organisms recovered	No. of mice	No. died	Days of death	Organisms recovered
10	1	14th	Culture sterile	10	7	2nd (3) 5th (2) 8th (2)	S. dublin 7999/39 var. B

Unvaccinated Controls

Inoculated Var. A				Inoculated Var. B			
No. of mice	No. died	Days of death	Organisms recovered	No. of mice	No. died	Days of death	Organisms recovered
10	1	10th	Culture sterile	10	7	5th (4) 12th (1) 15th (2)	S. dublin 7999/39 var. B

Further Virulence Tests.—As the avirulence of var. A was a matter that required some confirmation, a further series of virulence tests were made. In this approximately 500,000 live organisms of each variant were inoculated intraperitoneally into 20 mice. The results are shown in Table 9.

TABLE 9

Second Series Virulence Tests S. dublin 7999/39 var. B

Approx. No. of Organisms in Inoculum	No. of Mice Inoculated	No. Died	Days of Death After Inoculation	Organisms Recovered	S.A. Reaction Against K. Sera K.1	K.6	K.7
500,000	20	10	4th (2) 7th (2) 8th (4) 9th (2)	7999/39 B	—	++	—

Second Series Virulence Tests S. dublin 7999/39 var. A

			4th (2) 6th (3)	7999/39	+ or ++		—
500,000	20	11	7th (4) 8th (2)	vars. A & B	—	—	

It will be noted that in the var. A series 11 out of 20 mice died within the test time of one month, and in the var. B series 10 out of 20 died. This heightened virulence of var. A can, I think, be explained by the slide agglutination

tests done on the liver cultures of each dead mouse. As each mouse died a culture was made from its liver on plain agar, as in the previous series. As the mice were in almost every instance freshly dead, the cultures were usually pure. Slide agglutination tests were done on the heavy initial needle-track growth and on ten well separated colonies. These showed quite a large proportion of var. B colonies in the mice inoculated with the var. A suspension. The initial needle-track growth agglutinated only slightly in the K.1 S. paratyphoid A somatic serum, while the number of colonies that failed to be agglutinated in this serum varied from three to eight out of ten per plate culture. In no case was a colony found which gave rapid and complete agglutination, i.e., what I have arbitrarily called a true A variant. Cultures made from the mice that died in the var. B series always remained true to type and never showed any tendency to be agglutinated in the K.1 serum. From these tests it can be assumed that in this case the var. A culture was not pure and contained a proportion of var. B organisms, or that it reverted to var. B in the animal body (probably the former), and this I have held to be the cause of its heightened virulence. This experiment was therefore repeated. Cultures of both variants were again repeatedly replated until typical colonies were found as shown by rapid or no agglutination in the K.1 S. paratyphi A serum. These were inoculated on to agar slopes and after 18 hours the growth was washed off with sterile saline and approximately 500,000 live organisms were inoculated intraperitoneally into 25 mice. After the last mouse had been done in each series a small drop of each variant was blown out from the syringe on to an agar plate and spread to test for viability and purity. Both of these cultures had retained their variant characteristics after 18 hours' incubation. The results of the virulence tests are given in Table 10.

TABLE 10

Third Series Virulence Tests S. dublin 7999/39 var. B

Approx. No. of Organisms in Inoculum	No. of Mice Inoculated	No. Died	Days of Death After Inoculation	Organisms Recovered	S.A. Reaction Against K. Sera K.1	K.6	K.7
500,000	25	19	4th (6) 6th (3) 7th (4) 10th (2) 11th (2) 11th (2)	S. dublin 7999/39 B	—	++	—

Third Series Virulence Tests S. dublin 7999/39 var. A

500,000	25	4	10th (2) 21st 14th	C. muris Pasteurella (a)	

(a) The two mice that died on the 10th day showed pneumonic lesions from which pasteurella were isolated. The one that died on the 21st day had a Corynebacterium muris abscess in the salivary(?) lymph gland. The other had no lesions and the cause of death could not be ascertained.

Cultures tested by slide agglutination test against unabsorbed sera.

Thus it has been shown on three separate occasions, involving in all the inoculation of 150 mice, that variants of this strain 7999/39 of S. dublin which are agglutinated in a heterologous serum and which presumably contain the XII antigen, are completely avirulent for mice, and that variants which do not possess this factor, and which presumably possess the pure IX complex, are of considerable virulence.

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Discussion.—As my protocols on the occurrence of these variants are bulky I do not intend to give them in detail, but will confine myself to general statements and conclusions. This paper covers the study of this type of somatic variation over a course of two years. Only one strain, S. dublin 7999/39, was studied in detail as regards virulence and serological analysis, but several strains of S. dublin, S. typhimurium and S. gallinarum were tested for its occurrence. In each case it was comparatively easy to demonstrate colonies which were predominantly A or B, but it required months of selective replating to get colonies which were agglutinated rapidly or which failed completely to be agglutinated in the K.1 S. paratyphi A somatic serum. Two variants which readily agglutinated in the latter serum were isolated from a mouse strain and a calf strain of S. typhimurium, and it was found that heavy suspensions of these could remove from it the agglutinins for S. dublin 7999/39 var. A when tested by slide agglutination method. From my observations on the three above species of Salmonella I conclude that it is quite possible that the occurrence of this non-specific factor or probable XII antigen is of a diphasic nature, and from the avirulence of variants which contained it in preponderance, its appearance may possibly indicate a pre-rough stage. Old laboratory strains of S. dublin, S. typhimurium and S. gallinarum were usually able to produce a much higher proportion of non-specific variants than were young recently isolated strains. Also, old broth cultures of S. dublin 7999/39 var. A always showed a much higher proportion of R or SR variants than did var. B cultures of the same age. The appearance of these variants follows the same general lines as specific and group phase flagellar variation, and var. B colonies may, on subcultivation, give rise to a var. A growth and vice versa, but the tendency as far as I could ascertain was more towards the occurrence of A variants from B variant colonies.

It can hardly be deduced from the experiments described in this paper that all strains of S. dublin with the pure IX complex will always prove so virulent, or that those with the non-specific factor will always prove avirulent, but I think it can safely be asserted that Kenya strains of the three Salmonella species examined do show a diphasic somatic variation, and that one phase, the A phase, has antigens shared by other Salmonellæ.

Somatic suspensions of S. dublin 7999/39 var. A always proved more agglutinable than var. B against K.6 S. enteritidis somatic serum, S. dublin 7999/39 var. B somatic and OH sera, and against its own O and OH sera (Table 7); so that, in agglutination tests for enterica it may possibly be that the most agglutinable strain does not give the most selective reading, if, as in this case, its higher agglutinability is also associated wth the presence of a non-specific factor.

This work was carried out at the Veterinary Research Laboratory, Kabete, Kenya.

REFERENCES:

(1) Kauffmann: *Circular of the International Salmonella Centre, Copenhagen;* dated November, 1939.
(2) Kauffmann: *Protocols accompanying sera sent by the International Salmonella Centre, Copenhagen;* dated May, 1939.

SOME PRELIMINARY OBSERVATIONS AND REMARKS ON LIMB LAMENESS IN THE RACING TRACK GREYHOUND

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HAVING been associated with the above company for the past five years, during which period upwards of 600 cases of lameness have been indexed and observed, I now find that a careful analysis of this data affords most interesting and enlightening reading. It is hoped that the generalised schematic plan that is about to be outlined will act as a basic framework, from which, in due course, it may be possible to discuss and enlarge upon each individual or type of lameness.

The cases under question are made up as follows:

N.F.L. 223	O.F.L. 162
N.H.L. 64	O.H.L. 104

Now before sub-dividing the above preliminary facts it should be borne in mind that the track greyhound is called upon to perform at comparatively regular intervals, *a definite and set task,* which with the possible exception of hurdling varies but very, very little, that task being the covering, in an anti-clockwise direction, of anything from 400 to 700 yards, together with the negotiating of usually four slight bends, in the shortest time possible. This is a most important and key point to remember always, and in my opinion is the only explanation of the remarkably constant and fixed injuries that are sustained by the individual limbs; as a matter of fact I would go even further and suggest, if hampering, bumping, checking, etc., could be eliminated, that toe, tendon (flexor), track leg and hock injuries would become almost 90 per cent. standardised.

These few remarks should make the study of Sub-Division 1 more interesting, and also partly explain why it is possible to restrict the susceptible lame areas to such a comparatively small number—namely, in the fore limbs—shoulder, knee, tendons (flexor), toes; in the hind limbs—muscles, track leg region, hock and toes. Injuries sustained between these designated areas are met with, but are so few, and in the majority of cases so gross, as to merit separate record.

No cases of lameness resulting from paralysis of the peripheral limb nerves have been encountered, which is rather interesting, considering that the subject is frequently performing a task, during which one would think that the nerves in question would necessarily at times be susceptible to at least a few of the accepted causes of such lameness—i.e., bruising, straining, stretching, or even direct injury.

SUB-DIVISION 1

N.F.L. 223

Shoulder	52
Knee or carpus ...	23
Tendons (flexor) ...	12
Toes	136

N.H.L. 64

Muscles	22
Track leg region	approx. 20 cases treated
Hock	1 (severe collision)
Toes	23

O.F.L. 162

Shoulder	59
Knee or carpus ...	31
Tendons (flexor) ...	9
Toes	63

O.H.L. 104

Muscles	56
Track leg region ...	0
Hock	31
Toes	17

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What do these figures reveal? Primarily, that by far the most commonly injured limb is the near fore leg, and this is strikingly evident as far as toe lameness is concerned. Why should this be so? I would like at this juncture to put forward my own theory as to the factors controlling this, but would first point out that the majority of injuries are always sustained when approaching, actually in or coming out of the bends, and are *not,* as some readers might think, the result of gross collisions and hamperings. Other important and salient facts revealed are—the great variation in the incidence of toe lameness about the individual limbs, the lack of flexor tendon injuries in the hind limbs, the almost 100 per cent. restriction of hock lameness to the O.H., and the location of fore track leg only to the near hind.

Speaking generally, I am of the firm belief that most injuries can be said to be due to a momentary phase of muscular inco-ordination, occasioned by some temporary interference with the nervous (impulse) cum muscular (reaction) synchronisation, that must be 100 per cent. perfect if the subject is to negotiate the bends satisfactorily. I think this is borne out by the repeatedly observed fact that lameness is comparatively less evident after severe than slight hampering, the possible explanation being that the severely hampered dog becomes immediately aware that something is wrong, and so keys himself up to withstand the additional strain, whereas the slightly hampered subject tries to carry on with but little or no nervo-muscular adjustment whatsoever.

For the purposes of expressing my theory re the comparatively large number of injuries sustained by the near fore leg, I would liken the racing greyhound to a motor car, with the drive behind and the steering in front. Now what happens when the subject is approaching a bend at speed? A slight and gradual turn must be effected, otherwise he will continue straight on. Now, whatever mechanism governs the alteration in individual limb stride or power that must necessarily take place to bring about the turn, it would appear that the near fore leg is the pivot or hinge upon which this turning mechanism mainly depends. Now taking for granted the accepted fact that more weight is placed on the inner aspect of the foot than the outer, an examination of Sub-Division 2 would seem to confirm this N.F.L. pivot theory, as it will be immediately observed that the bulk of the toe injuries are located about the two innermost toes N.F.F., and it can here be stated without a shadow of doubt that by far the most commonly injured individual toe (or for that matter the most frequently injured unit as a whole) in the racing track greyhound is the innermost weight bearing toe—N.F.F.

That the other limbs do, and must, assist during the turn is common sense, and to further this argument it is of interest to record that of the total number of toe injuries on each foot, approximately 50 per cent. are partial dislocations (exception O.H., 33⅓ per cent.)—outward on the near side and inward on the off side. Bearing in mind the direction of the pull of gravity when taking a left-hand bend, these outward and inward dislocations sustained respectively by the near and off side limbs are what one would expect to find, but it is noteworthy that their occurrence should be so constant. Naturally, it is appreciated that the structures above the foot must play the major part in effecting the turn,

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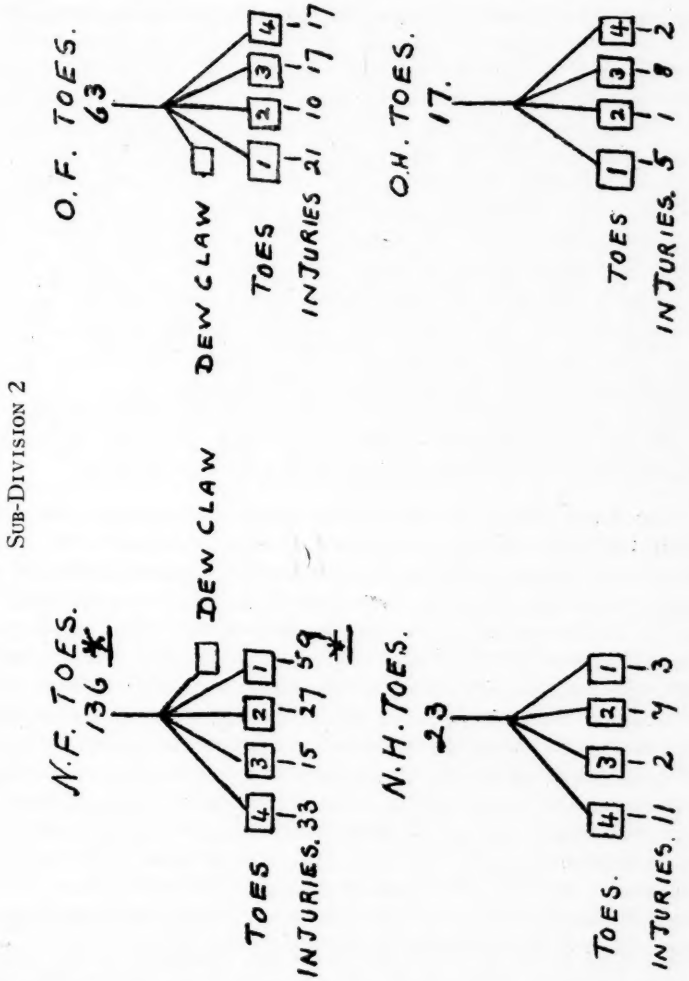
but these type dislocations sustained by that part of the limb that makes contact with the ground, together with the fact that the injuries on the off side are generally more severe (e.g., ruptured muscles, fractured toes, fractured hock) than on the near side, in my mind does give one an idea of what part the individual limbs are playing when cornering, and definitely infers that those on



the off side have to fulfil out outward jabbing cum holding in function as well as that of propulsion. Injuries to the dew claws are not included in Sub-Division 2, as the resultant lameness is usually of such a minor character as to merit but slight comment.

They are, however, quite frequently injured—the injury comprising a bruised or strained interphalangeal joint or, more commonly, a split, partial or

complete shelling of the attached nail. It is, however, very important that they should be recognised immediately and treated, as a split, partially or completely shelled nail appears to be susceptible very rapidly to infection, the treatment for the resultant and now quite pronounced lameness becoming extended from a few up to anything from seven to fourteen days. A point of interest here! Actual nail injuries about the weight-bearing digits are distinctly uncommon, and yet this vestigial remains of a once full functioning digit, particularly on the off side, is quite frequently injured. Why? Also apart from actual injury, immediately after a race the off dew claw is quite frequently found to be encrusted with hard earth and bits of grass, just as if it had gouged itself into the turf, the inference being that it is being brought into play. Now one realises that this would be the case when the dog pulls up at the "trip," but the predominance of off dew claw injuries and these earth encrustations suggest that it makes contact with the ground during as well as at the end of the race —presumably at the bends. A confirmatory note here—dew claw injuries are often sustained when the dog is being put through his "in-training" exercises, i.e., when he is having a free lance gallop in the open, and the usual history is that the subject let out a yelp when turning. In the majority of cases the injury is found to be on the opposite side to which the subject was turning, namely, right-hand turn—left dew claw injury, and vice versa. If the pictures that I have of greyhounds moving at speed are accurate and true, these injuries could easily be sustained both on the straight and the bends, as the apparent backward extension about the carpus must be seen to be believed.

I raise these points because quite a number of owners ask why these apparently useless claws are not removed at birth, and I feel that in the case of the racing greyhound we should be quite sure of ourselves before we take the easy way out and say: "Oh! it doesn't really matter, they aren't used at all." Personally, I have recently come to the decision that they would be better removed, as I quite fail to see how such a small structure could possibly play any important function during or at the end of the race—whereas frequently their presence has been proved to be detrimental, by virtue of the apparent ease with which they are injured. Furthermore, it should be remembered that they are indirectly connected with the knee by means of the small first metacarpal bone, the upper articular extremity of which forms a carpo-metacarpal union, which in itself is part and parcel of the whole knee joint proper. It is easy to visualise how in some cases these dew claw injuries could, and even might, be the precursor or exciting factor which at some later date causes actual knee lameness. If this is so, I feel that this is yet another and possibly a more important reason why they should be removed early in life.

I have purposely dwelt more fully on the injuries sustained by the limb parts that do, and can, make contact with the ground (i.e., toes and dew claws), because they more easily present a visible and diagnostic inference or guide as to the part played by the individual limbs, and I have tried to prove and confirm how cornering necessitates this different limb function, and why and how it is that the majority of injuries are sustained about the bends.

These facts lead on to more interesting material for investigation, as, for

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example, if we know the function of each muscle group or individual muscle (by virtue of their origins and insertions) and what the whole limb is trying to do, this, together with the recorded fact that muscle injuries, like all other injuries, are comparatively fixed and constant, should enable us to work out and possibly name the actual injured muscle, or muscle group, and thereby to be more confident in the assessment of the pros and cons of the said injury. This principle could more or less be applied to quite a number of injuries.

Some will probably say: well, what does it matter how or why certain injuries are sustained as long as you appreciate the site and extent of the injury and can prescribe an appropriate treatment! But a point here, which in my mind is of as much importance as the actual treatment proper. Many lay people can, and do, treat greyhound injuries, but they have not the least idea when the subject is again ready for progressive and intensive toning-up exercise, which is essential prior to any subsequent trial or race. I rank the assessment of this important phase in the disabled non-racing period as being on a par with the actual treatment proper, and it is of paramount importance that we should have a rough idea of its commencement and duration, relative to the type and degree of injury sustained. Just walking or trotting a dog up, at some unspecified period after his disablement, finding him sound and putting him out for trial or race is absolutely hopeless and just courting further trouble. Nevertheless, it is perfectly amazing how so many greyhound men, and even trainers, regard apparent visible soundness at the walk or trot, post injury, as being sufficient to merit the release of the dog for immediate trial or race, with the invariable result of either a breakdown of the old injury or, what is worse but just as frequent, a fresh injury about some previously sound limb. It has been noted that any new injury is usually located on the opposite side, more often the diagonal opposite, presumably as a result of the primarily injured but improperly restored limb not being able to fulfil its whole function. How can one attempt to overcome such an undesirable course of events? Only by having a knowledge of what I have been trying to explain—i.e., appreciating the difference in individual limb function, why or how certain areas or units are injured and the degree of the injury. Let's take an example—a simple injury to the first interdigital joint of No. 1 weight-bearing toe N.F.F. Now the actual treatment will be of short duration and much the same as for a similar injury about any other toe, but the treatment, plus the toning-up exercise, should exceed that of other similar toe injuries, because, knowing that this is the most commonly injured toe, it is logical to assume that it is either being used more or undergoing more strain, and is consequently more susceptible to a secondary breakdown.

All this has an important bearing on what sort of initial post-injury trial the subject should receive, namely, a short or a full distance. Personally, I am all in favour of increasing the toning-up exercise period and giving a full distance trial, as contrasted with a reduced toning-up period cum a sprint or pipe-opener trial, as I have found the latter to be more conducive to secondary breakdowns. There are a few exceptions to this rule, the majority of which are associated with post-disease trials.

Some remarks now re the detection of lameness. The importance of making

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an accurate diagnosis as to the location and degree of the injury as soon as possible after unsoundness cannot be over-emphasised. This is not possible with all injuries, as there are instances where, despite a poorly run race (which in your mind is definitely due to injury), one can either not spot the limb or can spot the limb but not the site. In such cases, however, lameness is positively evidenced within a few up to 24 hours, and is usually in the fore limbs—knee or flexor tendons—in the hind limb—hock. Both metacarpal and metatarsal phalangeal injuries at times present immediate diagnostic difficulties, but manipulation usually reveals their presence.

Why is it imperative to establish an early diagnosis? Because in a large number of cases, I should say over 50 per cent., the animal will, despite no treatment, be walking visibly sound within a few days, but actually he is not sound for the purpose for which he is required—i.e., trial or race—and if released for same will either finish very lame as a result of an aggravation of the primarily injured but undetected limb or, what is worse, will sustain an injury in a previously sound limb. A new injury may be only slight—a short period of treatment plus exercise may apparently cure the subject, who is again released for trial, only to go lame in the first undetected lame limb, the second detected lame limb or another previously sound limb. A subject like this rapidly goes from bad to worse, and in a period of a few months becomes a chronic, in other words, a useless racing proposition for either company or private owner—and, in my opinion, the origin of his successive injuries can be traced to that primary slight but undiagnosed first limb lameness.

A few miscellaneous points. It will be observed very quickly that lameness in the dog differs greatly from that in the horse. I must admit that at present I have not the necessary equine knowledge or facts to formulate any true comparison, but I do remember that the teaching at college was that at least half the causes of lameness in the horse were in the fore limbs—in the foot; in the hind limbs—the hock. Of course, this covered all types and breeds, and a true comparison here should only be taken from the racehorse. Nevertheless it would indeed be very interesting if those members who are, or were, attached to racehorses could either confirm or disprove the fact re the comparatively constant and fixed injuries that are sustained by the individual limbs of a subject that is frequently performing a definite and set task.

I would suggest that quite a large part of the material discussed in this article also draws attention to a non-veterinary but nevertheless very important factor in greyhound racing, namely, the construction of the running track, particularly the bends, and it would behove those people who are at some later date entertaining the projection of fresh tracks, or the improvement of existing ones, to pay great attention to this important but not infrequently overlooked matter. In concluding, I would like to say that the substance of the material discussed has been put progressively into practice at my track, and I can definitely state that the results all round have been equally progressive and satisfactory.

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FOLLICULAR MANGE. A THERAPEUTIC AGENT WHICH WARRANTS A TRIAL

By C. WENTWORTH ELAM, M.R.C.V.S., D.V.H.,
Liverpool University Veterinary School

A FEW years ago a client entered my office to discuss a business matter. Within a few minutes of his entry I became uncomfortably aware of a peculiar odour. Knowing the client well, I tactfully ventured to mention the occurrence, and he informed me that he was taking doses of Saccus alii as a medicinal agent. The odour was so marked that it appeared to be escaping from every pore of my client's body.

At that time I was treating two cases of follicular mange and had tried every suggested scientific remedy without success. The thought occurred to me that since it was almost impossible to find a specific external remedy which would penetrate the skin and destroy all the parasites in their "hide-outs," why not attack them from the "rear" by giving medicinal doses of this agent until the habitat of the parasites became obnoxious and untenable; in fact, it was quite possible that the odour might lower the morale of the parasites, even destroy them. The following day I commenced treatment of these two cases by administering 1 oz. doses of Saccus alii t.i.d. After a period of three weeks I noticed a marked improvement, and in three months' time all trace of the disease had gone.

Since that date I have treated a number of similar cases with Saccus alii (or the extract in capsule form), with the same encouraging results. I recall one case where the entire body, including the head, legs and feet, were in such an advanced state of disease that the case did not warrant even a trial with any form of treatment, but in twelve months time the dog had a perfect coat and showed no evidence of the disease. I would be interested to know the results of this treatment if any practitioner feels inclined to give this agent a trial. But practitioners have little time to go into details, etc.; perhaps our research masters could inform us the maximum doses dogs can tolerate and so shorten the period of administration when treating a case.

Abstracts

BRUCELLOSIS IN INDIA*

By J. B. POLDING
Research Officer, Imperial Veterinary Research Institute, Mukteswar

INVESTIGATION of contagious abortion in India has been the subject of an Imperial Council of Agricultural Research scheme for a period of over three years, and it has been observed that Brucellosis is by far the most common cause of this condition in Indian bovines. This article, therefore, has been written with the object of indicating what has so far been achieved in the study of this disease as it occurs in the Indian sub-continent.

The investigation of Brucellosis in India, however, is not yet complete, and

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in any case there is no limit to the amount of information that it seems desirable to acquire concerning so widespread a disease. In order, therefore, that Indian veterinarians may have readily to hand as much instruction as possible on the diagnosis and control of the disease, a series of appendices describing some useful technical methods have been prepared and are obtainable from the Director, Imperial Veterinary Research Institute, Mukteswar—Kumaun, U.P.

The Bacterial Subspecies Responsible for Brucellosis in India

The genus Brucella is usually considered to contain three variants, or subspecies, viz., *Brucella abortus, Brucella melitensis* and *Brucella suis;* nevertheless variants occur which are intermediate between any of these types. The host of predilection of *Br. abortus* is the bovine, of *Br. melitensis* the goat and of *Br. suis* the pig. Occasionally these hosts may be interchanged and all variants may at times occur in almost any other animal, including man.

Up to the present time, 46 Indian Brucella strains have been secured by the writer. Of these, 22 have been typed as pure *abortus;* 18 as intermediate between *abortus* and *melitensis;* and three as *melitensis.* The remainder were either lost before typing or have not yet been typed. Table I shows the hosts of origin and the character of these strains. It is noticeable that only three *melitensis* strains and no *suis* strains have been encountered; and that of the three *melitensis* strains, one came from man, one from a goat and only one from a cow. It is also to be observed that all four village strains are typed as *abortus-melitensis,** from which it is tentatively suggested that the indigenous Indian Brucella strain is of this kind. It may also be seen from the table that organised dairy farms are about equally infected with this type and the imported English type *Br. abortus.*

TABLE I

Indian Brucella strains

Host	No. of strains isolated	Typed as A	A/M	M
Dairy cow	20	11	8	1
Village cow	4	0	4	0
Buffalo (dairy) ...	16	10	6	0
Goat	1	0	0	1
Horse	1	1	0	0
Man	1	0	0	1
Total ...	43	22	18	3

A = abortus; A/M = abortus/melitensis; M = melitensis.

It is not usually feasible to distinguish the variant responsible for an outbreak of Brucellosis without first isolating the strain involved. The more strains that can be procured, therefore, the greater will be the amount of information available on the character of Indian infections.

The Susceptibility of Indian Stock to Brucellosis

As has been previously stated, almost all the higher mammals can on

*The term *abortus-melitensis* refers to an intermediate strain, exhibiting some characteristics of *Br. abortus* and some of *Br. melitensis.*

important animals are cattle, sheep and goats, the following remarks are confined occasion become infected with Brucellosis; but, as in India the commercially only to these species. Nevertheless, because observations are almost always influenced and obscured by other factors, such as climate and animal husbandry, it is difficult at present to give reliable figures illustrating the relative susceptibility of even these animals. On the whole, however, the conditions in military dairies and such like farms do not differ widely throughout Northern India; hence a statement drawn from an aggregate of these is not unworthy of consideration. Again, although it is possible that their climate is on the whole less likely to favour the spread of Brucellosis, the Punjab Grantee farms also merit consideration, as they are run on a system similar to that of the military dairies and they are probably the best available source of information on pure indigenous cattle. The following figures relating to such farms are abstracted from the records of this Institute:

TABLE II

The relation between the incidence of Brucella reactors and the occurrence of European blood in Military Dairy Farms

Military Dairy	Percentage of crossbreds in herd	Percentage of Brucella reactors
Allahabad	90	50
Ambala	82	45
Quetta	80	39
Lahore	17	24
Ferozepore	1	13
	—	—
Average	54	34

TABLE III

The relation between the incidence of abortion and the occurrence of European blood in Military Dairy Farms

Military Dairy	Percentage of crossbreds in herd	Percentage of aborters		
		All breeds	Cross breeds	Indigenous breeds
Allahabad	90	42	45	20
Ambala	82	25	27	16
Quetta	80	30	33	17
Lahore	17	10	4	11
Ferozepore	1	10	0	10
	—	—	—	—
Average ...	54	22	21.5	15

The term "aborters" refers to animals with a history of abortion present in the herd at the time of a single random observation.

The figures in Tables II and III tend to show that the higher the proportion of European blood in a herd, the higher is the incidence of infection in all classes of animals.

TABLE IV

The percentage of aborters at a single random observation in the classes of stock indicated

Class of animal	No. of animals observed	Percentage of aborters
Crossbred stock of 15 military dairy farms ...	1,333	16.0
Country-bred stock of 13 military dairy farms ...	164	7.5
Buffaloes of 15 military dairy farms	1,880	5.0
Indigenous cows not in contact with crossbred cattle (Northern India)	4,495	3.5
Buffaloes not in contact with crossbred cattle (Northern India)	895	3.5
Buffaloes in special environments (1 farm) ...	281	15.5
Indigenous cows in special environments (3 farms)	502	3.5

(Northern India is taken to be the territory north of a line drawn from Bombay to Calcutta, Bengal and Assam excluded.)

From Table IV it will be seen that: (*i*) the largest number of abortions occur in the crossbred stock of military dairies, (*ii*) the rate of abortion in the country-bred stock of military dairies is about half of that in crossbred stock, (*iii*) the abortion rate in the buffaloes of military dairies is rather less than that in the country-bred stock in the same establishments, (*iv*) the abortion rate is about equal in country-bred stock and buffaloes when neither is in contact with crossbred stock, but, at the same time, is less than that in the same sort of stock when it is cohabiting with crossbred animals. The last two categories listed in Table IV require some discussion. The special environments mentioned are the conditions of animal husbandry in and around Bombay. These conditions will be discussed more fully in a later section, and it is sufficient to say here that buffaloes in this environment show a startling increase in the rate of abortion as compared with the rate in the Punjab, whilst cows in the same environment show the same rate as for Northern India. The numbers recorded in these classes are small, especially in the case of buffaloes, as figures are difficult to obtain from many Bombay farms. Nevertheless, many additional farms have been examined, and in the absence of records the general impression received supports the figures shown in the table. It is concluded, therefore, that when buffaloes are removed from their natural environment they become much more susceptible to Brucellosis than similarly moved indigenous cattle. Indeed, in these circumstances, their susceptibility perhaps reaches or even surpasses that of the crossbred cows of Northern India.

There is some evidence to show that either the susceptibility of Indian cattle is decreasing or that better methods of animal husbandry are limiting the spread of Brucellosis. Table III was constructed from records made during the period 1924 to 1929, and Table IV from figures collected from 1939 onwards. It is noteworthy that the average number of abortions in the former table is greater than that in the latter. Again, the annual reports of the military dairy farm show a progressive decrease in abortions from all causes during the years 1932 to 1939. Lastly, in 1923, the mean number of abortions present at a single random observation on five Punjab Grantee farms was 3 per cent. of the 1,264

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animals observed, while at the time of the writer's visits in 1939 this figure had fallen to 1 per cent. of 835 animals. If this decrease is indeed due to a reduction in susceptibility, it may only represent a phasic decrease, but if it is due to better animal husbandry then the decrease is likely to be permanent.

The susceptibility of various classes of stock to repetitions of abortion is shown in Table V.

TABLE V

The likelihood of repeated abortions in various classes of Indian stock

Class of stock	Percentage of animals aborting				Average expectation of loss of calves per aborter	Population observed
	Once	Twice	Thrice	Four times		
Crossbred cows	79.5	17.5	2.5	0.5	1.24	198
Country-bred cows	90.0	9.0	1.0	0.0	1.11	75
Buffaloes ...	90.0	10.0	0.0	0.0	1.10	91

Assuming that the percentage abortions shown in Table IV are correct, then amongst 100 crossbred cattle 16 may be expected to be aborters and their total loss of calves would be $16 \times 1.24 = 19.84$ calves. The same figures for country-bred cows and buffaloes work out at 8.33 and 5.5, respectively, and by making the last figure unity, the preceding figures would be 1.5 for country-bred cows, and 3.5 for crossbred cows. Thus, susceptibility to abortion amongst buffaloes, country-bred cows and crossbred cows is 1 : 1.5 : 3.5. Applying the same calculation to buffaloes in special environments, their susceptibility figure works out at a little over 3.

The susceptibility of sheep and goats to the Brucella types extant in India appears at the present time to be small, but the final word has yet to be said on this subject.

CAN PHENOTHIAZINE BECOME TOXIC FOR HORSE AFTER PASSAGE THROUGH PIGS ?*

By T. LLOYD JONES and J. A. KELSO

THE following is the summary of an interesting observation :—

Four horses died within seventy-two hours from the time they were seen eating straw at a manure pile. The manure had been thrown from a pig pen, in which nine pigs were treated with 160 grams of phenothiazine mixed in a thick grain slop one week previously. Two of the horses died within twenty-four hours from the time the manure was thrown from the pen. Inco-ordination of movement and diarrhœa were shown by all of the horses and purple-coloured urine had been voided in the yard. Phenothiazine was obtained from the stomach contents of one of these animals.

The history of these fatalities gave no incriminating evidence that was not related to the eating of straw at a manure pile containing the fæces of pigs treated with phenothiazine.

* *Can. J. Comp. Med.*, 1943, vii, 240.

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TOXICITY OF RED SQUILL FOR SWINE AND RATS*

By R. GWATKIN and P. J. G. PLUMMER

FOLLOWING the carrying out of experimental work, the authors' summary is :—

Nine pigs were given powdered red squills in doses of 50, 100, 200, 300 and 400 milligrams per kilogram of body weight. The first two doses did not produce symptoms, but 200 mg./kg. proved toxic. One of two pigs that received 300 mg./kg. died, as did the male and female that received 400 mg./kg.

Sixty-four white rats were given doses of powdered squill ranging from 50 to 800 mg./kg. Thirty-four males and thirty females were employed. Ten males (29.4 per cent.) and fourteen females (46.6 per cent.) died. In the groups receiving the largest dose (800 mg./kg.) the susceptibility was reversed, as 83.3 per cent. of the males and 50 per cent. of the females died. However, the numbers were small, consisting of six and four rats respectively.

A mixture of one ounce of squill to one pound of chop was not eaten by six pigs to which it was offered. The same dose mixed with ground meat was refused by two pigs to which it was offered.

Lesions in rats and swine poisoned by red squill were extensive and somewhat inconsistent, not only in the two species but in individuals. Less tissue damage occurred when very large doses were given, because death intervened before severe cellular changes occurred.

It may be fairly concluded from these experiments that powdered red squill is as toxic for pigs as rats, but that the former are not likely to eat baits containing one ounce of squill to one pound of grain or meat. However, the possibility of their doing so cannot be excluded.

* *Can. J. Comp. Med.*, 1943, vii, 244.

Obituary

GEOFFREY HERBERT LIVESEY

IT is with deep regret that we have to record in this, the first number of THE VETERINARY JOURNAL in the New Year, the passing of G. H. Livesey, who died on November 30, 1943. By his death the veterinary profession has lost one of its finest and greatest members. His career has been told elsewhere; suffice is it for us to write of him as we knew him. We never knew him in his days as a veterinary practitioner: we knew him, however, in connection with his other activities as a veterinary surgeon. His interest in all matters pertaining to the welfare of the veterinary profession and to individual members of the veterinary profession stands out clearly and distinctly in our mind. Many are there who have cause to bless the name of Livesey for his timely help and encouragement in their moments of depression. Those of us who had the privilege of associating with him in some of the aspects of the work in which he interested himself saw that fine side of his character and nature which made the man and enabled him to lead his very full life. To many of us his passing

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has left a gap which we shall find difficulty in filling and an empty place which will not be easily taken by anyone else.

Livesey's work for the veterinary profession will continue to live. His interest in the rising generation was amply displayed by his actions on behalf of the training of veterinary students. He realised the great need for a fundamentally sound scientific education for the preparation of young men and women for a career in veterinary medicine: his views on the subject were well known and his efforts to enable them to obtain the most satisfactory form of education will, we feel assured, have their own reward. We were privileged to work with him on this subject and came to realise how very much he had it at his heart. His presence at Council and Committee meetings was eagerly sought, and his speeches and contributions were indicative of the thought he gave to the education of the younger generation.

He had many activities, most of which were associated with his profession. His work for N.A.R.P.A.C. was typical of his interest in the welfare of animals in general, and his efforts for the proposed R.C.V.S. Bill clearly demonstrated his attitude towards bringing the veterinary profession into its proper sphere in all things relating to the welfare of animals.

One could write much more of the qualities of this outstanding man. This note may, however, be sufficient to record our deep appreciation of all he did for his profession, to pay a tribute to a great man and to hope that his life may inspire all of us to do our bit, however small it may be, to carry forward the work he did.

MAJOR-GENERAL W. S. ANTHONY, C.B., C.M.G.

Major-General Anthony died on November 15 after a professional life spent almost entirely in the Army. Beginning as a Veterinary Lieutenant in 1895, the year after he qualified from the Royal Veterinary College, he rose to the rank of Major-General in 1929, when he was appointed Director-General Army Veterinary Services at the War Office. He finally retired in 1933. During the Great War he held posts in India, Mesopotamia and at home. For his services he was awarded various honours, including the C.B. and C.M.G., and was appointed Colonel Commandant of the Royal Army Veterinary Corps in 1934.

We cannot do better than quote from *The Times* of November 23, 1943, in which reference was made to him:—

"The death of William Anthony has caused a real sense of loss to those whom he served so loyally and to whom he gave so much. He added to professional knowledge of a remarkable quality a rare judgment and originality. It was natural that he should reach the highest post in his profession. But it was the other sides of his character which added so much to his attraction. He was deeply read and added to a profound religious faith a touch of mysticism inherited from his Welsh forbears. Wherever he served he always strove to know the people. He understood and was loved by Irish peasants, and by a strange, almost kin-like collection of horse dealers, jockeys and vagrants in Baluchistan. His wealth of quaint and original anecdote will be sadly missed."

XUM

GEORGE DUNLOP MARTIN

On November 27, 1943, there passed away a well-known and beloved veterinary practitioner—Captain G. Dunlop Martin. Although over 70 years of age, he retained his activity till the end and died practically in harness. He served in the Royal Army Veterinary Corps during the last war and returned to practice on demobilisation. His interest lay mainly in small animals, for the treatment of whose ailments he was held in high esteem. His contributions on various aspects of small animal work have appeared in the veterinary literature, including this journal.

Dunlop Martin was also interested in other branches of the veterinary profession, for we find him as President of the Section of Comparative Medicine of the Royal Society of Medicine in 1941; during his year of office many different subjects formed the programmes.

We shall always remember him by his unassuming, quiet and gracious manner, and his memory will be dear to those who knew him well.

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